SEPTEMBER 1956

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Disease-a-Month

Surgery in Heart Disease

LEWIS DEXTER

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Surgery in Heart Disease

LEWIS DEXTER

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Lewis Dexter

received his M.D. degree from Harvard Medical School, interned in medicine at the Presbyterian Hospital, New York City, was a Research Fellow under Dr. Soma Weiss at the Boston City Hospital and the Peter Bent Brigham Hospital, and spent a year in Buenos Aires, Argentina, working in physiology under the direction of Dr. Bernardo A. Houssay. Since 1941 he has been a member of the faculty at the Harvard Medical School, where he is now Assistant Professor and Tutor in Medicine. He is on the staff at the Peter Bent Brigham Hospital, where he is now Physician. At present his chief research interests are in cardiovascular disease.

AN EVALUATION of the present status of cardiac surgery is difficult because of the speed with which the field is progressing; new technics of evaluation are being devised and refined, and reports of new surgical experiences are appearing almost daily. The purpose of this report is to try to evaluate the surgical procedures as they are currently being employed.

HISTORICAL BACKGROUND

The surgical approach to heart disease is not a new development, early attempts having been introduced half a century ago. These early attempts, however, were doomed to failure because the related fields of thoracic surgery and anesthesia had not developed sufficiently to allow the surgeon to open the chest with impunity. Surgery of the heart had to await the development of thoracic surgery, accomplished during the 1920's and 1930's. As soon as the chest could be entered surgically with facility, cardiac surgery was attempted. Thus Schmieden, in 1926, performed pericardiectomy for constrictive pericarditis, an operation which, with certain modifications, has been carried out ever since. Gross, in 1937, ligated the patent ductus arteriosus. Crafoord and Gross, in 1945, independently devised a method for the correction of coarctation of the aorta by resection of the narrowed area; Blalock and Taussig, in 1945, anastomosed the subclavian artery to the

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pulmonary artery in order to increase pulmonary blood flow in the tetralogy of Fallot. These were all extracardiac procedures. The heart itself remained immune to surgical attack.

The era of what might be termed "blind intracardiac" surgery may be considered to have started in the 1940's. World War II led to the successful removal of bullets and shrapnel fragments which had become lodged in the heart muscle and within the cardiac chambers and great vessels of men in combat (Harken). After the war, surgeons naturally turned their attention to intracardiac surgery, from which stemmed operations devised for the relief of mitral stenosis (Bailey, Harken), pulmonic stenosis (Brock), atrial septal defect (Murray), aortic stenosis (Bailey), and several rare types of congenital heart disease. All these surgical approaches were blind, in the sense that the surgical procedure was carried out through the sense of touch but not of direct vision.

Within the past few years, open heart surgery has begun to be performed successfully. Direct vision surgery has been made possible by the use of hypothermia or by a circulatory by-pass of the heart of one sort or another.

Hypothermia consists of lowering the body temperature to approximately 30° C by placing the anesthetized patient in a tub of ice-water, and at this temperature it is usually safe to occlude the circulation for periods up to 10 minutes, during which time the heart may be opened, operative correction carried out, and the cardiac incision closed. In this way, Lewis and Tauffic performed open heart surgery for atrial septal defect and Swan for pulmonic stenosis. The advantages of the use of hypothermia are obvious, but there are two important disadvantages: (1) The intracardiac surgical procedure must be accomplished within a short period of time. This of necessity restricts the use of hypothermia to simple procedures. Correction of complicated lesions is not satisfactory. (2) All tissues except the heart tolerate hypothermia well. Arrhythmias appear at low body temperatures, and these are difficult to control and introduce an important factor of surgical risk over and above that of the surgical procedure itself.

Circulatory by-passes have been used by Gibbon, Dennis, Dodrill, Kirklin, and Lillehei. Problems concerned with the development of a cardiac by-pass have been concerned chiefly with a method for the aeration of blood with proper control of oxygenation, CO_2 concentration, and pH and avoidance of hemolysis. These and many other allied problems have been

the subject of considerable investigation and account for the slowness of development of a practical procedure for by-passing the heart and insuring a relatively dry field during the operative procedure. Lillehei and his associates have tried to simplify the technic, (1) by using normal human donors as a source of arterialized blood by means of cross-circulation, (2) by use of a reservoir of arterialized human blood and, more recently, (3) by use of a pump-oxygenator of simple design and practicality. Lillehei, utilizing these circulatory by-passes, has already been able to perform open heart surgery in man.

From the foregoing, it is apparent that this field of cardiac surgery has developed rapidly and is in a state of change at this very moment. Progress is being made at such a rate that many of the statements to follow will have to be revised within the year. The era of open heart surgery has already arrived and promises to take on widespread application within the near future. Many lesions which are now considered to be inoperable promise to become operable. Statistics relative to surgical risk and surgical effectiveness will have to be revised in the light of these newer developments. The discussion to follow is an attempt to evaluate surgery as it is today from the point of view of an internist. Particular emphasis will be placed on its effectiveness and risks and on the problem of selection of patients for surgical therapy.

Only the main types of heart disease for which surgery is available will be discussed in any detail, and the vast bibliography will of necessity be restricted by the limitations of space. I hope the authors whose references have been excluded will forgive their omission.

CONSTRICTIVE PERICARDITIS

This was the first cardiac disorder to be corrected surgically with a consistently high degree of success. It has been well described in the literature. It is not common. It consists of a fibrous encasement of the heart. Although the fibrotic process usually involves the whole surface of the heart, including the A-V grooves and the great veins and arteries adjacent to the heart, it is extremely rare for constriction to reach such magnitude as to obstruct the flow of blood into and out of the heart. This author has yet to observe a pressure gradient across these areas, although it certainly must rarely occur. Rather, it is a cardiac constriction pressing on the heart so as

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to raise the diastolic pressure in all chambers together with a corresponding rise of systemic and pulmonary venous pressures.

Constrictive pericarditis is due to tuberculosis, pyogenic pericarditis and, in about half of most reported series, to unknown causes. There is a strong suspicion that the unknown group are tuberculous, but definite proof is lacking.

Clinical manifestations are largely those of cardiac tamponade. The first symptoms are easy tiring, a noncongestive type of exertional dyspnea (the "restricted ambulation" of Evans and Jackson), edema and, later, ascites and hydrothorax. Appetite becomes impaired; the patient becomes cachectic and incapacitated by fluid accumulation which becomes increasingly refractory to medical therapy.

The first physical sign to appear is distention of veins from the increased venous pressure. The heart does not shift as the patient changes position; heart sounds become more distant. Gallop rhythm is common; blood pressure tends to be low, pulse pressure to be narrow, and a paradoxical pulse appears.

On x-ray examination the heart is usually not greatly enlarged, if at all; its pulsations are classically reduced fluoroscopically or by roentgenkymography. Calcification of the pericardium occurs in somewhat less than half the cases. Low voltage with flat or inverted T-waves is revealed in the electrocardiogram. Angiocardiography may show a wide space between the opaque material in the cardiac chambers and the outer cardiac shadow, indicating either a thick pericardium (constrictive pericarditis) or endocardium (endocardial fibroelastosis).

Several characteristic but unfortunately not diagnostic findings are found by the cardiac catheter. First, the tip of the catheter does not abut the cardiac border as viewed fluoroscopically. This is similar in implication to the findings by angiocardiography. Secondly, the pulse pressure in the right ventricle is narrow, usually being less than the diastolic pressure. Thirdly, there is a plateau of all diastolic pressures, practically identical values being found in diastole in systemic veins, right atrium, right ventricle, pulmonary artery, pulmonary "capillary" and presumably left atrium and left ventricle. These are in turn equal to the pericardial pressure in experimental pericardial effusion and are therefore presumptively the same as the intrapericardial pressure in constrictive pericarditis. Fourthly, the atrial pressure curve is M-shaped, with the a and v waves representing the two peaks.

The mean pressure is elevated. Fifthly, the right ventricular pressure has a diastolic dip followed by a rapid rise to a plateau. Unfortunately, all these findings are found also in diffuse myocardial fibrosis and endocardial fibroelastosis, and have been reported in amyloidosis of the heart.

The prognosis is poor. Although there is occasionally a cessation of progression of disability, in general there is a progressively incapacitating downhill course culminating in death.

Initially surgery consisted of making a window in the pericardium to decompress the heart. Through this window, the surgeon attempted to obtain a cleavage plane manually between pericardium and epicardium (pericardiolysis). Most patients were improved by this procedure, but many had to be subjected to reoperation. In recent years, Holman has advocated radical resection of the pericardium, including that not only overlying both ventricles but over the atria and great veins as well. As noted above, anatomic involvement of the great veins, auricles, and A-V grooves usually exists, but physiologic obstruction to blood flow in these regions, as indicated by pressure recordings, must be extremely rare. Resection of the pericardium from these thin-walled structures must increase the risk of surgery and does not appear to this author to be necessary.

The risk of surgery varies, of course, with the pathologic process encountered, the age of the patient, and the severity of disability, but in round numbers it approximates 10 to 15%.

Even though patients usually obtain considerable benefit from surgical removal of the pericardium, it is the rare patient who has complete restoration to normal, even after extensive resection of the pericardium. The resting venous pressure is almost routinely somewhat elevated and rises higher on exercise. Patients rarely resume as active a life physically as they did before the occurrence of their pericardial disease but they do resume a much more active one than preoperatively. Evidence indicates that constrictive pericarditis is more than a pericardial disease. At postmortem, the process is often found to have infiltrated the myocardium, which contains areas of inflammatory reaction and varying degrees of fibrosis. Muscle fibers are often atrophic. From the appearance of the patchy but often widespread involvement of the myocardium, it seems probable that not only is the strength of myocardial contractility im-

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paired but the normal co-ordination and efficiency of myocardial contraction for ejection of blood is seriously interfered with. In addition to residual heart disease, restrictive pleuritis, from involvement of neighboring structures by the inflammatory process, not infrequently produces marked respiratory disability.

Although the above remarks may appear discouraging, actually this is effective surgery and restores most patients to a useful life. Perhaps a third remain fairly limited.

In recent years Holman and other surgeons have emphasized the importance of early diagnosis and surgical intervention. It is practically impossible, however, to make an early diagnosis of constrictive pericarditis because the first manifestations are those of tamponade, by which time the pericardial disease is already far advanced. Pericardial resection at the time of the initial pericarditis with effusion due to tuberculosis or a pyogenic infection has therefore been advocated on the basis that in many cases constrictive pericarditis will eventuate, that repeated pericardial tapping is not without risk, that the longer the pericardial inflammation continues the greater the likelihood of myocardial involvement, that pericardiectomy is technically much easier to perform during the effusive than during the constrictive phase, and that the use of antibiotics removes the risk of spread of the tuberculous process to adjacent structures. Even though this approach to the problem appears radical to conservative medical men, there is much truth to the thesis of early surgical intervention when the etiology is tuberculosis or a pyogenic organism. The so-called benign, relapsing idiopathic pericarditis is, however, benign as its name implies and probably never produces pericardial constriction. Pericardial resection was performed in one such case at the Peter Bent Brigham Hospital with the thought that a recurrence of pericarditis might be prevented. There being no pericardium, the patient of course had no more "pericarditis," but he did have several more episodes of fever and chest pain identical with those prior to pericardiectomy. Therefore, there appears to be no rationale for pericardiectomy in nontuberculous and nonpyogenic forms of acute pericarditis.

There is no question that early surgical intervention is indicated for tuberculous and pyogenic pericarditis, but whether it is desirable to perform pericardiectomy as early as the effusive stage remains to be seen. The likelihood of the development of constrictive pericarditis subsequently

must be weighed against the operative risk and long-term effectiveness. Such statistics are not yet available. In the experience of one group, however, thoracotomy for acute cardiac tamponade carries the same mortality as aspiration but a somewhat higher morbidity.

PATENT DUCTUS ARTERIOSUS

This abnormality consists of a persistence of the fetal structure which in intra-uterine life allows blood to pass from the pulmonary artery to the aorta, thus by-passing the lungs. As soon as the lungs expand after delivery, this structure ceases to serve a useful purpose. Over the course of days or weeks, it becomes obliterated. In some cases it persists, for reasons largely unknown. There are numerous excellent descriptions of this disease. As pressure in the pulmonary artery falls below that in the aorta, a shunt of blood flows from aorta to pulmonary artery, on through the lung to the left atrium, left ventricle, and aorta. Thus, the left ventricle pumps more blood than the right and is put under stress the magnitude of which depends on the size of the ductus and of the shunt. If the shunt is of sufficient size, the above structures pumping or conducting the shunt appear prominent on x-ray examination. The shunts may vary in volume from a trickle to as great as perhaps 15 L. per minute, in which case the ductus is as large as a person's thumb.

Only with the larger ductuses does the left ventricle fail. The right ventricle, which does not participate in the shunting, remains unaffected. The ECG shows, if anything, left ventricular hypertrophy.

Uncomplicated patent ductus arteriosus usually presents a fairly straightforward clinical picture characterized by full activity in those with a small ductus, varying degrees of disability in those with larger ones, a continuous murmur in the pulmonic area, absence of cyanosis, a tendency to widening of the pulse pressure, an ECG without evidence of right ventricular hypertrophy, and an x-ray showing enlargement of the left ventricle and of the pulmonary vascular markings. In most instances ordinary clinical methods suffice to make an accurate diagnosis. Occasionally the typical murmur may not be present in infancy and early childhood, when the difference in pressure between the aorta and pulmonary artery is not great and the flow through the ductus is correspondingly small. Although certain patients with patent ductus

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arteriosus may live to a ripe old age, the average life span is about 35 years, due to the occurrence of heart failure in 30% and subacute bacterial endocarditis (in the pre-antibiotic era) in about 40%. Subacute bacterial endocarditis may occur in the ductus itself or more commonly on the wall of the pulmonary artery opposite the orifice of the ductus. Although subacute bacterial endocarditis is currently considered to be a curable form of heart disease, it must be remembered that it still is serious and carries about a 20% mortality.

Other complications can be retardation of physical development, pulmonary hypertension and, rarely, rupture of the pulmonary artery.

The question of surgery for the uncomplicated, asymptomatic patent ductus arteriosus depends on several factors. Operation is curative, a normal heart and circulation resulting. There is no resultant predisposition to subacute bacterial endocarditis. The operation consists either of ligation of the ductus or division, with the majority of experienced surgeons preferring the latter because, although it is technically more difficult, it does not carry a higher risk, it is more definitive, and recanalization almost never occurs. In the most experienced hands, the uncomplicated ductus bears a surgical risk of less than 3%. Decision with respect to surgery must take into account the dangers of subacute bacterial endocarditis and the other complications mentioned above. With a 40% over-all incidence of subacute bacterial endocarditis and a mortality of 20% of these, it can be seen that these patients run about an 8% risk on nonsurgical management. Since the operative risk is less than the nonoperative, it is this author's opinion that surgical eradication of this lesion is indicated in all uncomplicated cases.

Occasionally, when a complicating subacute bacterial endocarditis is unresponsive to antibiotic therapy, closure of the ductus is life-saving in that the infection then becomes responsive to the antibiotics. This fact has been well documented and there is a real place for surgery during active subacute bacterial endocarditis refractory to antibiotic therapy.

The optimal age for surgery is in late childhood or adolescence. If the lesion causes disability, operation may be life-saving during infancy. Surgery should rarely be considered after the age of 35 or 40 because any cardiac disability which then presents itself is usually due to some serious associated

heart disease, for example, hypertension or coronary disease.

THE SO-CALLED "ATYPICAL DUCTUS." In a small number of cases, pulmonary hypertension occurs out of all proportion to the pulmonary flow. Calculation of the pulmonary vascular resistance reveals it to be elevated. The clinical picture is atypical for patent ductus arteriosus and resembles that of Eisenmenger's syndrome. Exertional dyspnea and fatigue are prominent symptoms. Angina or anginoid pain may occur on exertion or even at rest. The continuous murmur is barely audible or absent, depending on the proximity of the pulmonary arterial pressure to that in the aorta. There is liable to be a harsh blowing diastolic murmur down the left sternal border. A systolic murmur is sometimes present in the pulmonic area. The pulmonic second sound is loud. Intermittent or constant cyanosis may be present. The cyanosis may be universal or confined to the lower extremities. Clubbing may be present. X-ray shows a left ventricle of normal size and enlargement of the right ventricle and pulmonary artery, and prominence of the pulmonary vascular markings. A hilar dance may be present. The ECG shows right ventricular hypertrophy. Cardiac catheterization may reveal some left-to-right shunt from aorta into pulmonary artery, severe pulmonary hypertension, and some arterial oxygen unsaturation. In three proved cases in our own experience, both systolic as well as diastolic pressures have been practically identical in the brachial and pulmonary arteries, both at rest and during exercise, beat for beat, indicating a wide-open communication between the aorta and pulmonary artery. In other instances, however, these various pressures are not identical. We have not seen both systolic and diastolic pressures identical at rest and during exercise, however, in any other condition, although theoretically it should occur in patients with a large aortic-pulmonary fenestration. In those who are cyanotic, a sample of blood from the femoral artery will contain less oxygen than one obtained simultaneously from the brachial artery. This latter finding is diagnostic of patent ductus arteriosus with a reversed shunt.

The nature of the pulmonary vascular disease in these cases appears to be due to more than one process. These have been recently reviewed by Hultgren *et al.*, who point out that four general possibilities exist: (1) It is the result of a prolonged increase in total pulmonary blood flow such as appears to occur in certain cases of atrial septal defect and

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ventricular septal defect with large pulmonary blood flows. (2) It is the result of multiple pulmonary emboli, since a number of cases quoted by Hultgren have shown the presence of recanalized and organized intraluminal thrombi at postmortem. (3) It is due to an antecedent acute pulmonary arteritis with subsequent healing and vascular occlusion. (4) It is due to the persistence of the high pulmonary vascular resistance of the fetus into postnatal life, conditioned by the presence of a free communication between aorta and pulmonary artery from birth. The main body of evidence indicates that this is the usual cause. With the exception of the experience in Mexico, those patients with severe pulmonary hypertension almost invariably have a ductus of very large size, some limitation of activity can be traced back to the early years of life, and the clinical course of these individuals is almost identical with that of patients with Eisenmenger's complex, or a large interventricular septal defect—i.e., all are lesions in which there is a free communication between both sides of the circulation at the level of the ventricles or of the great vessels. Although limited all their lives, such patients may develop no serious incapacity until the third or fourth decade. The high pulmonary vascular resistance found in many cases in Mexico City irrespective of the size of the ductus raises the question of the synergistic action of hypoxia (elevation 7,400 feet) and increased flow through the lung as a cause for the common occurrence of pulmonary vascular disease. Their experiences seem to indicate that the pulmonary vascular process is at least in part an acquired rather than a congenital lesion.

The almost universal experience has been that surgery in patients with a severe degree of pulmonary hypertension has a high risk, particularly when the pressures in the two circuits are essentially equal or when there is a right-to-left shunt. There is no large series of cases from which to state the precise risk, but the figures available indicate the surgical risk to be about 50% when the pressures in both circuits are approximately the same and the shunt is from right to left. If, on the other hand, there is a left-to-right shunt and the pulmonary arterial systolic pressure is below that in the aorta, there appears to be a place for surgery, even though the risk would appear to be fairly high and the regression of the pulmonary vascular disease after surgery is irregular and inconstant. It is generally believed that if, on clamping the

ductus, pressure in the pulmonary artery rises, division or ligation of the ductus should not be performed. If the pressure falls, surgery should proceed. Thus the decision with respect to surgery must take into account the prognosis, which, in general terms, will be good perhaps to the age of 25 to 30 on the average, as opposed to a very high surgical risk no matter when the surgery is performed.

AORTIC SEPTAL DEFECT

In its pathologic physiology, this lesion is similar to the patent ductus arteriosus except that the connection between aorta and pulmonary artery is located just above the level of the aortic and pulmonary valves. These are not common lesions and they are frequently inoperable. A few, however, have been surgically obliterated. Whether or not they are amenable to surgery depends on the size of the communication and its distance from the coronary ostia. They may be indistinguishable preoperatively, from patent ductus arteriosus.

ATRIAL SEPTAL DEFECT

Atrial septal defects have come under increasing study in the past few years. The defects may occur high in the septum, often associated with anomalous pulmonary venous drainage, or in the middle, or low. They may be single or multiple, small or large. The septum may be practically absent. Their embryologic development and precise anatomy have been described in detail, particularly as they bear on surgical correction.

From the surgical viewpoint, there are two fundamentally different types of defect—those which are in the middle and upper part of the septum and those in the lower part of the septum adjacent to the A-V valves, the ostium primum type of defect. The latter defect may involve and interfere with the function of the mitral and tricuspid valves, and there may be a defect of the ventricular septum adjacent to the A-V valves, the so-called auriculo-ventricularis communis.

Defects of the atrial septum result in an abnormal connection between the two sides of the heart. If these are small (less than 1 sq. cm.) there is a small, clinically unimportant shunt of blood from left atrium to right atrium. If it is much larger than 1 sq. cm., it results in a large left-to-right shunt—

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in fact the largest shunts that are seen occur in this condition.

The left-to-right shunt can be calculated to be as high as 15 or 20 L./min. or more. This shunt goes from left atrium to right atrium, right ventricle, through the lung, and back to the left atrium. A heavy work load is thus placed on the right ventricle, which becomes dilated and hypertrophied. The flow of blood from the common atrium through the mitral valve into the left ventricle and aorta appears to be reduced below normal in many of these individuals, thus probably accounting for the common complaint of easy tiring and, in childhood, the occasional retardation of growth. The left ventricle is characteristically small and hypoplastic. The left-to-right shunt itself appears to be innocuous clinically for a period of years. Beginning about the third decade, however, changes in the pulmonary vasculature sometimes appear and pulmonary hypertension ensues, due in part to pulmonary vascular changes and in part and perhaps more importantly to thrombosis. The right ventricle fails to empty itself with each systolic ejection so that the left-to-right shunt becomes progressively reduced. From our studies and those of Swan *et al.* a right-to-left shunt is largely a mixing problem. Unoxygenated systemic venous blood and oxygenated pulmonary venous blood enter the common auricle and become mixed to a certain extent. In the presence of a large left-to-right shunt, a right-to-left shunt is either absent or else is so small as to be of no clinical significance. When the left-to-right shunt becomes reduced so that right and left ventricular outputs approximate one another, there is a better opportunity for interatrial mixing and a right-to-left shunt becomes more pronounced with the appearance of cyanosis—the *cyanose tardive* of atrial septal defect. Usually, however, this occurs only after the right ventricle has failed as a result of pulmonary vascular disease.

The hypoplastic left ventricle appears to function adequately until some added disease process affects it, and then appears to fail more easily than normal. Hypertension, mitral regurgitation and, in the older age group (over 40), degenerative heart disease, are the commonest factors. Defects of the ostium primum producing mitral regurgitation and the ventricular septal defect of an A-V communis put an undue load on the left ventricle. In any event, when the left ventricle fails, its output, already low, does not and cannot fall much further, but its diastolic pressure rises; since there is

a free communication between both auricles, both ventricles, and both venous systems in diastole, the pressures in all these areas rise correspondingly. An elevated systemic venous pressure in these patients is indicative of left ventricular failure—not right ventricular failure.

Patients with atrial septal defect may have no disability for many years. Some, however, are retarded in growth in childhood, have repeated respiratory infections, or tire easily. *Cyanose tardive* and pulmonary vascular complications appear most often in the third and fourth decades of life. Many patients have reduced energy and easy fatigability all their lives and, for reasons not clear, these symptoms may suddenly become accentuated to such a degree as to interfere seriously with life's responsibilities. Dyspnea is common but it is of the noncongestive variety. There is simply shortness of breath on activity which normally produces no breathlessness, but there is no sense of air hunger, apprehension and suffocation so characteristic of congestive dyspnea. True congestive dyspnea occurs only in those with an associated left ventricular failure or, rarely, severe mitral stenosis (Lutembacher's syndrome).

The average life span of patients with atrial septal defect is 36 or 37 years. In the series of Burrett and White, 54% survived beyond the age of 40. Patients may live into the eighth decade of life.

Physical examination may reveal a gracile habitus with arachnodactyly, a heart of normal or enlarged size, enlargement in the region of the main pulmonary artery, a systolic murmur of moderate intensity in the third or fourth left intercostal space. An apical diastolic rumble is not infrequent, and only rarely indicates mitral stenosis. The pulmonic second sound is accentuated and widely split.

The ECG reveals changes of right ventricular hypertrophy in all but the mildest cases and of incomplete or complete right bundle branch block in about 90% of most reported series.

Fluoroscopy reveals enlargement of the right ventricle, pulmonary artery, and hilar and intrapulmonary vascular markings. A hilar dance is common. The left atrium, ventricle and aorta are normal or reduced in size.

Cardiac catheterization reveals the entrance of arterialized blood into the right atrium. The catheter may often be directed through the defect into the left atrium and ventricle.

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Pulmonary vascular disease may be recognized, not by measuring pulmonary arterial pressure alone, but by relating pressure to flow:

$$PVR = \frac{PAm - LAm}{CO} \times 1332$$

where PVR = pulmonary vascular resistance, dynes seconds cm.$^{-5}$
PAm = pulmonary arterial mean pressure, mm. Hg.
LAm = left atrial mean pressure, mm. Hg. In large atrial septal defects, the right atrial mean pressure may be used, since the difference in pressure between left and right atria is negligible.
CO = cardiac output, cc./second.
1332 = 1.36 (specific gravity of mercury) × 980 (gravity factor).

A high flow alone raises the pulmonary arterial pressure and it is only by calculating resistance by this formula that pulmonary vascular disease can be identified.

Lutembacher's syndrome is the name applied to the coexistence of severe mitral stenosis and atrial septal defect. Minor degrees of scarring of the mitral valve are not uncommon, but severe mitral stenosis is rare. This is difficult to recognize clinically because a typical mitral stenotic murmur frequently occurs in the absence of mitral stenosis. Pressure in the left atrium rises as the mitral valve narrows and the same elevation occurs in the right atrium, right ventricle, and both venous circuits. Left ventricular failure results in similar pressure rises in the same chambers. History, physical examination, fluoroscopy, and electrocardiography fail to differentiate left ventricular failure from mitral stenosis in these patients. Diagnosis depends on cardiac catheterization, with measurement of pressures on each side of the mitral valve. A higher pressure in the left atrium than in the left ventricle in diastole is diagnostic of mitral stenosis, and to my knowledge is the only certain method of identifying this lesion, short of exploratory cardiotomy or postmortem examination. Mitral stenosis of clinical significance can be dismissed if the systemic venous pressure is normal.

Partial anomalous drainage of the pulmonary veins into the right atrium occurs frequently in association with atrial septal defect and in a sense is a natural occurrence when the atrial defect is high on the atrial septum. It produces an added left-to-right shunt and an exaggeration of the clinical manifestations of atrial septal defect. Its recognition is difficult clinically and by cardiac catheterization. Swan *et al.*

have utilized the dye dilution technic for accurate preoperative recognition.

An *ostium primum* defect is a more serious lesion, both clinically and surgically, than defects of the ostium secundum and patency of the foramen ovale. It may be suspected when the systolic murmur is maximal in the fourth and fifth left intercostal space. It may furthermore be suspected when there is co-existing evidence of mitral regurgitation, as indicated by an apical pansystolic murmur of different character than that along the left sternal border, a systolic pulsation in the jugular pulse, or systolic expansile pulsation of the liver.

Atrio-ventricularis communis is an even more serious lesion, because in addition to an ostium primum type of defect there is a low interventricular septal defect. A left-to-right shunt occurs at both the atrial and ventricular levels. The association with mongolism is frequent.

The clinical course is marked by a progressive downward course, culminating in congestive heart failure. If the defects are small, the lesions are fairly well tolerated, but if large, poorly so. According to Rogers and Edwards 70% of patients are dead by the age of 2 and only 9.4% survive to 30. Cyanosis is rare and, when it does occur, it is usually on the basis of such large defects that the lesion merges with that of a cor biloculare.

On physical examination, there is often a harsh systolic murmur and thrill, maximal in the third and fourth left intercostal spaces. Electrocardiography and fluoroscopy usually show the changes of an isolated atrial septal defect, but, in addition, in some cases, there is evidence of enlargement of the left ventricle. Regurgitation of the A-V valves may be evident. The cardiac catheter may pass from right atrium to left atrium, right ventricle to left ventricle, or right atrium to left ventricle.

Surgery of atrial septal defect has developed rapidly in the past eight years. Surgical correction may be divided into methods of blind closure and more recently those of closure under direct vision in the open heart.

In 1948, Murray placed mattress sutures approximately in the plane of the atrial septum from the site of the transverse sinus behind the aorta to a position posteriorly between the superior vena cava and the right pulmonary veins. When the sutures were drawn taut, the anterior and posterior walls of the atria were compressed together. Swan inverted both right and left atrial appendages into the septal opening. This tech-

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nic was later abandoned because the defect was not always closed and there was a danger of partially obstructing the orifices of the pulmonary veins. Bailey sutured the right atrial wall into the septal defect by means of purse-string sutures guided by a finger within the heart (atrio-septopexy). Hufnagel and Gillespie described the use of double plastic buttons to close the defect which were shown to have little clinical application because of their tendency to dislodge. Gross *et al.* described the technic of using a rubber dam (well) sutured to the right atrium through which the defect was closed by direct suture or by means of sheets of polyethylene. Söndergaard and Björk and Crafoord described their technic of circular silk suture through the septum.

With these various technics, the main problem is that of closing the defect completely. If a defect as small as 1.5 to 2 cm.^{2} persists, the shunt will remain as large as ever and the patient will receive no benefit. Swan recently reviewed the pertinent literature of blind atrial defect surgery and pointed out that the over-all surgical risk was 29% and that in about 1 case in 5 the defect was not satisfactorily closed. These results are undoubtedly a little more gloomy than is actually the case because they include the surgeons' earliest experience as well as patients with ostium primum defects and patients with severe pulmonary vascular disease. A more realistic figure would be considerably lower, perhaps 10%, but there is still too small a number of patients operated on for objective evaluation of the surgical risk and the incidence of defect closure by these methods.

Direct vision surgery has become possible through the use of hypothermia and of an extracorporeal circulation. Lewis *et al.* and Swan and his colleagues have utilized hypothermia as an aid to direct vision surgery. Their combined experience of surgical repair of defects of the ostium primum and secundum includes 62 patients with a 16% mortality and a failure of complete closure in 4 (1 in 16).

The problem of correction of a secundum defect is much simpler than that of a primum. Although primum defects have been closed, the risk at this writing is over 60% and most surgeons wisely "back out" without attempting their correction. The problem is that surgery may injure the conduction system or the mitral valve. Scar formation from the sutures may result in heart block a week or more after the operation.

It would appear that correction of an ostium primum de-

fect will eventually be best accomplished under direct vision. The same is true of atrio-ventricularis communis. Although blind closure has been done, direct vision would appear to be almost mandatory. Using cross-circulation with direct vision, Lillehei and co-workers have operated on two patients with A-V communis. The ventricular and atrial defects were closed by suture and the valvular deformity was corrected. One patient died; the other survived and was considered to be cured.

The indications for surgical intervention for atrial septal defect are not too clear because, as indicated above, the surgical risk is not negligible, the results are not consistently good, surgical technics are improving rapidly, and many patients will do well for many years. On the other hand, by the time the patient is cyanotic and truly symptomatic, pulmonary vascular disease probably is present and then the surgical risk is exorbitant. Fortunately, this complication is rare before the third decade and only spotty in its occurrence thereafter. Although many patients remain asymptomatic for decades, others fail to grow and to develop normally, have repeated respiratory infections, or tire to such an extent that their daily activities are seriously limited. Any of these symptoms is considered proper indication for surgery. Another indication is cardiac enlargement or a heart that is becoming larger, irrespective of symptoms. Some advocate surgery simply because by cardiac catheterization the right ventricular output is three times the left ventricular. This finding is practically a *carte blanche* indication, because it is present in almost all defects of any size. Until atrial defect surgery approaches the risk and effectiveness of patent ductus arteriosus surgery, such a generous selection seems hardly justified. It would seem more proper to reserve surgery for those with clinical evidence of disability.

There is perhaps one exception to this rule; i.e., early pulmonary vascular disease. An increased blood flow through the lung, so characteristic of this condition, necessitates an increased pressure. A pulmonary systolic hypertension up to about 40 mm. Hg. can be on a flow basis alone. The calculated pulmonary vascular resistance is equal to the ratio of pressure to flow (see equation on p. 16) and is either normal or low in atrial septal defect. In the course of time it may rise with associated changes in the pulmonary vasculature and especially widespread thrombosis. In the presence of a pulmonary vascular resistance about five times normal or

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more, the risk of surgery is prohibitive. Just how high the pulmonary vascular resistance can be before surgery is contraindicated remains to be determined.

VENTRICULAR SEPTAL DEFECT

Ventricular septal defects are not the benign lesions that textbooks would lead one to believe. Only the smallest holes are benign. The vast majority are not tiny and eventually lead to disability. The clinical picture of ventricular septal defect varies considerably.

When a communication exists between the two ventricles, cardiovascular adjustments may reside in the defect itself or in the small pulmonary blood vesesls. When the defect is small, the opening produces enough resistance to flow to allow pressures, flows, and resistances in each circulation (pulmonary and systemic) to be normal. When there is a free communication between the two ventricles, circulatory adjustments depend on responses in the small pulmonary blood vessels. A free communication implies equalization of systolic pressures in both ventricles, pulmonary artery, and aorta. A balanced flow with little or no shunt occurs when the pulmonary vascular resistance and the systemic vascular resistance are identical. If the pulmonary vascular resistance is less than the systemic, the pulmonary blood flow will exceed the systemic as blood shunts from left to right, and vice versa. Some infants with a congenital ventricular septal defect die of the effects of a large left-to-right shunt with pulmonary edema and hemorrhage.

The high resistance in the pulmonary vasculature may be due to a persistence of the fetal form of vasculature with its thick muscularis and narrow lumen and its continuance may be due to the high pressure in the pulmonary circuit. Later in life intimal proliferation usually appears. Whether the high resistance in the lung is due to the histologic lesions seen under the microscope or to vasoconstriction or both remains to be determined. In any event, there is increasing evidence to indicate that following a normal birth, these patients may do poorly for a few years, perhaps due to a sizable left-to-right shunt as Damman suggests, and then improve as the shunt is reduced by an increase of the pulmonary vascular resistance. Along about the third to fourth decade, these patients may have a reversal of their shunt with cyanosis associated with the development of intimal changes in the

pulmonary vasculature and a higher resistance in the pulmonary circuit than in the systemic.

The clinical course depends on the size of the defect and the pulmonary vascular resistance. Small defects produce little or no disability. Larger ones may lead to death in infancy and early childhood as a result of heart failure from a large left-to-right shunt; the child may be "sickly" for a few years and then improve; or almost full activity may be enjoyed from birth until the third or fourth decade, when cyanosis may appear, at first on effort and then constantly, due to a right-to-left shunt. Symptoms then consist of increasing dyspnea, anginoid type of pain, easy fatigability, occasional hemoptysis, and ventricular irritability with episodes of paroxysmal ventricular tachycardia. Death may occur as a result of heart failure or it may be sudden—presumably on the basis of ventricular fibrillation.

On physical examination, there may or may not be cyanosis and clubbing. There is a systolic murmur and often a thrill maximal at the third and fourth left intercostal spaces, an accentuated P_2 and occasionally a pulmonic diastolic murmur.

The ECG reveals right ventricular hypertrophy if there is a free communication between the two ventricles, but with small defects without right ventricular hypertension the ECG is usually balanced.

Fluoroscopy of those with small defects reveals a heart that is not enlarged; both ventricles are of normal size, the pulmonary artery and vasculature are within normal limits. With larger defects and increasing pressure or flow in the right ventricle and pulmonary circuit, there is more and more enlargement of the right ventricle, pulmonary artery, and pulmonary vasculature. The left atrium is normal in size. The left ventricle is enlarged only in those with large left-to-right shunts.

Cardiac catheterization is of value diagnostically in revealing (1) the pressures in the right heart and pulmonary artery, (2) a left-to-right shunt at the ventricular level, and (3) the magnitude of the pulmonary vascular resistance.

Small ventricular defects are well tolerated and are compatible with full activity and a long life. It is difficult to give a figure for prognosis in patients with larger defects, which would include Eisenmenger's complex and cor triloculare biatriatum. There is a wide individual variation in outlook, some patients dying in infancy and some as late as the

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seventh decade. In general, however, the average life span is perhaps 25 years.

Murray attempted closure of ventricular defects by passing fascia lata strips in an anteroposterior direction through the ventricular septum so as to include the defect. Bailey used transventricular pericardial grafts in an effort to close the defect. Lillehei *et al.* introduced direct vision repair of ventricular defects, using controlled cross-circulation from a human donor, and reported their results in 22 patients. The defects were closed by suture. There were 7 deaths and 15 recoveries. Three patients had been recatheterized postoperatively at the time of the report and there was no longer any left-to-right shunt. A subsequent note described the experience in 25 patients with ventricular septal defect. There were 7 deaths (6 in infants). All patients had been seriously ill and had pulmonary hypertension averaging 75% of the systemic pressure. Five had been catheterized 4 to 9 months later. There were no ventricular shunts in any, and in 3 the pulmonary arterial pressure had become normal. Kay *et al.* reported their results in 6 patients, 2 repaired by closed technic and 4 under direct vision. There were 3 deaths. Kirklin and his collaborators, using a Gibbon-type extracorporeal circulation, have repaired defects of the ventricular septum under direct vision in 20 patients in most of whom the pulmonary arterial pressure approximated that in the aorta. There were 4 deaths and in the 16 survivors there were no abnormal shunts, as indicated by dye curves.

Closed technics for the closure of ventricular defects have met with little success, and direct vision would appear to be essential. Experience with direct vision closure is too meager for evaluation except to say that closure is possible, the surgical risk, though high at the moment, promises to become lower, and pulmonary hypertension disappears at least in some cases.

TETRALOGY OF FALLOT

The tetralogy of Fallot consists of (1) pulmonic stenosis, (2) overriding of the aorta, (3) interventricular septal defect, and (4) right ventricular hypertrophy.

The pulmonic stenosis may vary from mild to atresia and it may be valvular or infundibular. From post-mortem findings Keith and Donzelot *et al.* reported the following frequency of types:

	Keith	Donzelot *et al.*
Pulmonary atresia	20%	18.5%
Valvular stenosis	10	7.5
Infundibular stenosis	48	44.5
Combined valvular and infundibular stenosis	22	29.5

Whether or not there is true anatomic dextroposition of the origin of the aorta is a matter of controversy. In most instances the root of the aorta is in its normal position but the defect in the membranous portion of the interventricular septum provides free communication between both ventricles and the aorta, resulting in physiologic as opposed to anatomic overriding.

The interventricular septal defect is always high and is usually of such size as to create a free communication between the left and right ventricles with identical pressures in each. Although anatomically the two ventricles are separate, physiologically they are common ventricles.

Right ventricular hypertrophy is a constant feature of the tetralogy of Fallot and is due to the high pressure in the right ventricle.

Pulmonary atresia (pseudotruncus) is manifest by a single fibrous cord in the position of the pulmonary artery. A hypoplastic pulmonary artery having a diameter of only 2–3 mm. may make surgical intervention difficult or impossible. In the usual case, the pulmonary artery is smaller than normal but in some it is of normal caliber and wall thickness. Finally, post-stenotic dilatation of the pulmonary artery and hypoplastic vessel walls may complicate surgical procedures.

In this condition, the venous return empties into the right atrium and right ventricle. This ventricle ejects blood at systemic pressure and there are two possible outlets—the pulmonary artery and aorta. The amount flowing to the lung is determined almost entirely by the degree of pulmonic stenosis, which, being characteristically severe in these cases, results in a diminished pulmonary blood flow. The amount passing from right ventricle to aorta (the right-to-left shunt) is determined largely by the total systemic resistance in relation to the fixed resistance at the pulmonic valve. If the systemic resistance is low, as for example after exercise, the right-to-left shunt is increased and the patient becomes more cyanotic. If the systemic resistance is increased, the right-to-left shunt becomes less. The left ventricle, having received the small flow from the lungs, ejects its contents into the

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aorta. This, plus the right-to-left shunt from the right ventricle, is likewise much lower than the normal flow. These are the principal features of the abnormal physiology. More detailed descriptions appear elsewhere.

As may easily be appreciated, this is a complicated lesion anatomically and physiologically which in different individuals embraces the whole range of mildness and severity of pulmonic stenosis, ventricular defect, and functional overriding of the aorta; moreover, other complicating defects may be present.

The clinical picture in the typical case is one of cyanosis and clubbing of the fingers, often at birth but sometimes not occurring for several months after birth. Dyspnea on effort, limitation of exertion, retarded growth and development, and cerebral disturbances in the form of headaches, convulsions, vertigo and coma are frequent. Squatting is a characteristic posture, assumed especially after quiet standing or walking. It is preceded by a fall of arterial oxygen saturation with prompt restoration on squatting. It is prevented by wrapping the legs with ace bandages. Several patients have volunteered the information that if they did not squat, they would faint. A variety of explanations have been offered for this phenomenon but no complete studies have been made of its physiology. It seems to occur in any situation associated with venous pooling in the lower extremities, with its associated decrease in systemic peripheral resistance which favors a right-to-left shunt and cyanosis. Its prompt relief by squatting appears to be somewhat analogous to the relief obtained in normal individuals who are about to faint.

Physical examination in the classical case reveals cyanosis and clubbing and, usually, physical underdevelopment but normal intellectual development; the heart is "boot-shaped"; there is a harsh pulmonic systolic murmur and thrill, maximal between the second and fourth left intercostal spaces in the majority of cases; the pulmonic second sound is usually diminished. The rhythm is regular.

The ECG shows right ventricular hypertrophy.

Fluoroscopy reveals a boot-shaped heart with a narrow waist in the region usually occupied by the pulmonary artery. The right ventricle is enlarged, the pulmonary vascular markings are decreased, the left atrium and the left ventricle are of normal size.

Secondary polycythemia may be extreme. Hematocrit readings of 85 are not rare. The total blood volume is in-

creased. The plasma volume is normal or slightly decreased. The red cell volume is markedly increased.

Angiocardiography is a valuable preoperative test. It reveals (1) simultaneous filling of the pulmonary artery and aorta, (2) the stenotic portion itself, (3) reduction of blood flow through the lungs, (4) the size of the pulmonary artery, (5) configuration of the great vessels of the neck. Each of these points is of concern to the surgeon.

Cardiac catheterization is a diagnostic test and reveals (1) the nature of the pulmonic stenosis—valvular or infundibular, (2) the existence of a ventricular defect or functional overriding of the aorta (equal systolic pressure in aorta and right ventricle, passage of the cardiac catheter from right ventricle into aorta), (3) the pulmonary and systemic blood flows, right-to-left shunt, and degree of arterial oxygen desaturation.

The tetralogy of Fallot is a serious lesion. The average age at death is 12 years. A few survive to adult life but may rarely live into the sixth decade. In most cases, however, life is one of semi- or complete invalidism.

There are four operative procedures for patients with the tetralogy of Fallot:

1) Blalock operation. Anastomosis of subclavian artery to pulmonary artery. The rationale of this procedure is to increase the blood flow through the lung, thereby increasing the amount of fully oxygenated blood returning to the left heart and systemic circuit.

2) Potts operation. This consists of a side-to-side anastomosis between the aorta and pulmonary artery, and has the same conceptual rationale as the Blalock operation.

3) Brock operation. This consists of transventricular relief of the pulmonic stenosis, on the basis that its relief will increase the pulmonary blood flow.

4) Eradication of all abnormalities (pulmonic stenosis, ventricular septal defect, and overriding of the aorta).

The surgical risk in 857 cases reported by Taussig and Bauersfeld was 15%. The surgical risk of the Potts type of operation is approximately 15% for children under 3 and 2.8 to 4.6% for those over 3. In 140 cases recently reported by Brock, valvulotomy was performed in 50 cases with a 12% mortality, infundibular resection in 62 cases with a 13% mortality, and combined valvulotomy and infundibular resection in 28 cases with a 7% mortality—an over-all mortality of 11.5%.

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Experience with the curative operation is still too meager for evaluation. Lillehei *et al.* have, however, reported 10 cases: one with pulmonary atresia and a ventricular septal defect, 1 pentology with valvular pulmonic stenosis, 7 tetralogies with infundibular stenosis, and 1 tetralogy with a valvular stenosis. There were 4 deaths and the 6 survivors were considered to be cured.

When classified as good, satisfactory, unchanged, or dead, with the Blalock and Brock type of procedure, the results are as follows:

	Taussig and Bauersfeld	Brock
Good result	78%	76 %
Satisfactory	4	10.5
No change	3	0.7
Died	15	11.5
Total number	857	140

By good result is meant that cyanosis disappears or is greatly improved, that physical activity is almost normal, arterial oxygen saturation rises between 75 and 90%, polycythemia regresses, and patients feel markedly improved. A satisfactory result indicates some improvement but a persistence of polycythemia.

The optimal time for surgery is between the ages of 5 and 10, since the risk is higher before and after these ages.

These various operative approaches to this disease are impressive in the results obtained. Surgery has been changing, however, in the direction of curing the lesion, first with Brock's direct relief of pulmonic stenosis and now with the beginnings of curative surgery under direct vision. To quote Sir Russell Brock, "I think the correct treatment for tetralogy must concern itself in relief of the pulmonic stenosis and in relief of the ventricular septal defect. That is what we have to progress toward . . ."

As a generalization, almost all of these patients are badly in need of surgery. Which type of operation is performed, however, is the surgeon's own decision which often cannot be made until the heart is exposed at operation.

TRANSPOSITION OF THE GREAT VESSELS

This lesion is one of the commonest causes of cyanosis in the newborn. The aorta, including the coronary ostia, arises from the right ventricle, the pulmonary artery from the left.

For the child to survive, there must be either an atrial or ventricular septal defect or a patent ductus arteriosus to allow mixing of venous and arterial blood.

Surgical attempts at correction have consisted of the following: (1) Creating an atrial septal defect and systemic-pulmonary artery anastomosis. This has met with only partial success. (2) Transposing the pulmonary venous return to the right atrium and the systemic venous return to the left atrium. This has been only partially successful; the coronary sinus drainage remains on the wrong side of the heart. (3) Transposing the great arterial trunks, but this leaves the coronary ostia on the wrong side of the circulation.

Mustard *et al.* have attempted to transpose the left coronary artery along with transposition of the great arterial trunks, but without success to date.

In summary, surgery for transposition is far from satisfactory at this time. It is to be hoped that as the technics of open heart surgery become better perfected, surgical correction of this lesion will become possible.

COARCTATION OF THE AORTA

Coarctation of the aorta consists of a narrowing of the aorta just distal to the origin of the left subclavian artery and ligament of the ductus arteriosus. An extensive collateral circulation develops between the internal mammary arteries and the epigastric branches of the external iliacs and the abdominal wall, between the scapular branches of the subclavians and the upper aortic intercostals on the thoracic wall, and between the superior intercostals within the chest. These collaterals may be visible and palpable particularly over the upper back, and they may also erode the ribs, producing a characteristic radiologic picture. The extent of the collateral circulation is directly proportional to the severity of the narrowing of the aorta. Coarctation is said to be 3 times more common in males than in females. Although symptoms may occur in childhood, they usually develop in early adult life and consist of palpitation, tinnitus, a feeling of warmth about the head, vigorous pulsations in the neck, coldness of the legs, numbness and pallor of the lower extremities, and even intermittent claudication. The diagnosis should be suspected in every case of hypertension in young people. The most important single clinical examination is a comparison of blood pressure in the arms and legs with blood

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pressure cuffs of the same size. Ordinarily the pressure thus recorded is higher in the legs than in the arms, whereas in coarctation of the aorta the reverse is true. Hyperactive pulsations in the upper extremities contrast with the diminished pulsations in the femoral arteries. The upper extremities feel warmer than the lower. The heart tends to become progressively enlarged, with the enlargement in the region of the left ventricle. A systolic murmur may be heard over the heart; it is loudest at the base and may be as loud or even louder over the back.

The ECG usually shows the changes of left ventricular hypertrophy.

The most important radiologic sign is erosion of the undersurfaces of the ribs, which may occur as early as 6 years. The heart may show some enlargement, particularly in the region of the left ventricle.

Angiocardiography is an extremely important procedure to use in delineating the precise anatomy of the coarctation, particularly its length and the size of the aorta, both proximal and distal to the narrowed area. This is best performed by injection of contrast material into the aorta above the coarctation through a catheter introduced either into the left brachial artery or one of the femoral arteries.

The clinical course is variable. Many patients are asymptomatic until middle life, but in general the earlier the symptoms appear, the worse the prognosis. The main complications are hypertension, left ventricular failure, rupture of congenital aneurysms of the circle of Willis, rupture of the aorta, and subacute bacterial endocarditis on the coarctation or on the commonly associated bicuspid aortic valve. Reifenstein, Levine, and Gross found the average age at death to be 30.5 years; 61% of cases did not survive their fortieth year. Death was caused by rupture of the aorta or from a cerebral hemorrhage in the second and third decades, bacterial endocarditis or aortitis in the first five decades, and congestive heart failure in the third to fifth decades.

Surgery for coarctation of the aorta was introduced by Crafoord and Nylin, and by Gross and Hufnagel in 1945. The technic consisted in resection of the narrowed area of the aorta, and end-to-end anastomosis of the cut ends. When, after resection of the coarcted area, the distance between the two cut ends is too great, it is necessary to use some other procedure than end-to-end anastomosis. Various technics have been employed, such as end-to-end anastomosis between

the left subclavian and the distal end of the aorta and end-to-side anastomosis between the subclavian artery and the distal portion of the aorta beyond the coarctation without resecting the coarctation itself. The most successful way of joining widely separated cut ends of the aorta is by the use of arterial homografts, first employed by Gross in 1949 and widely used by others since then. Although such grafts become fibrous, they remain strong and tough. They endothelialize and have shown but little tendency to aneurysm formation in ensuing years. The main problem in the use of arterial homografts is the shortage of supply. Many attempts have been made to use prosthetic materials to re-establish continuity and many substances are being investigated. It would appear at this time that the use of synthetic materials is still in the experimental stage although their use appears promising.

The surgical risk in 342 cases collected from the literature in 1952 by Donzelot *et al.* was 14%. The results of operation are good in the vast majority of cases. In 71% of the 100 cases reported by Gross in 1950 all symptoms disappeared. Hypertension disappeared in 80% of the cases for a short time after surgery but no long-term follow-up figures are available. In 29% the results were variable.

In considering the indications for surgery, the relatively poor statistical prognosis without operation must be balanced against an operative risk of about 14%, a cure rate that still remains to be determined, and marked improvement in about 70%. The ideal age for operative intervention is between 8 and 20. This is a time when, in the average case, the patient has few if any symptoms. Delay of surgery beyond this age raises the operative risk. In infancy, coarctation of the aorta is one of the main causes for congestive heart failure. In recent years operative correction of coarctation of the aorta in infancy has been performed as a life-saving procedure. It is the opinion of this author that all cases of coarctation up to the age of 30 or 35 should be seriously considered as operative candidates. This is particularly true if there are any symptoms or signs of circulatory abnormality.

AORTIC ANEURYSM

A description of the clinical manifestations of aortic aneurysms and of peripheral occlusive vascular disease will not be made here because the subject is too vast and they cannot

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actually be classified as cardiac surgery. Aneurysms occupy an important place in the field of vascular surgery, however, because of the striking advances that have been made in the use of arterial homografts and the stimulus that it provides to discover suitable synthetic prostheses or substitutes for the vascular areas to be resected.

Aneurysms frequently rupture, with a resultant fatal hemorrhage. Medical therapy has little to offer beyond the symptomatic. Surgical attempts at control or obliteration have had a long and varied history. This has recently been summarized by Mahorner who traced the historical development of aneurysmal surgery from the second century A.D. to the present. Ada and West describe the recent operative technics that have been employed: (1) Ligation, (2) intrasaccular wiring, (3) wrapping of aneurysm with fibrogenic materials, (4) lateral aortorrhaphy, (5) excision and use of arterial graft, (6) temporary by-pass or shunt around the excised segment to avoid cord ischemia and damage during prolonged complicated grafting procedures, and (7) controlled hypothermia to prevent ischemic damage to the cord during surgery.

Excision of aortic aneurysms is feasible and the continuity of the aorta can be satisfactorily restored. The immediate benefits are impressive. Long-term follow-up studies are of course still not available. To quote Mahorner, "There are few technical achievements in surgery more brilliant than the conquest of aortic aneurysms by excision of the involved segment and replacement with a homologous graft. Like so many advances in surgery, the method of its accomplishment lay for a long time written and recorded for all eyes to see* before it was applied deliberately and successfully in an attack on this distressing malady which by its magnitude seemed to forestall thought and aggressive action."

It is hardly possible to evaluate the risk of this type of surgery for two reasons: (1) The majority of patients are in the older age group and as such are not as good risks as those of younger age; (2) the risk will vary with the extent and location of the excision. Surgical excision and restoration of the arch of the aorta is obviously a more formidable procedure than excision and restoration of a small aneurysm in the descending aorta. That it is feasible is evident. The overall risk as reported by Cooley, DeBakey, and Creech was 22%

*Refers to the work of Alexis Carrel who between 1907 and 1912 replaced segments of the aorta of dogs with homologous grafts for which, together with allied work, he received the Nobel Prize.

for their first 50 cases and 14% in the next 51, which included a higher proportion of poor risk cases.

The earlier types of surgery for aortic aneurysm have been increasingly replaced in the last few years by excision of the lesion and restoration of continuity, especially by the use of homografts and vascular substitutes. Although it is too early to state with certainty, this type of surgery appears to be curative.

STENOTIC VALVE LESIONS

Stenotic valve lesions will be considered only as mechanical obstructions to blood flow, since it is the surgical relief of this mechanical obstruction that results in benefit to the patient. Stenosis of the four valves must be considered separately because each produces its own type of disability and each poses a different surgical problem. The main problem of surgery is relief of the stenosis without producing regurgitation. Except for the pulmonic valve, substitution of regurgitation for relief of stenosis is without benefit to the patient.

Only a slight increase in orifice size results in striking clinical improvement. The reason for this is explained physiologically by the relation between valve size, which the surgeon attempts to enlarge, and pressure and flow, which are immediately responsible for the patient's disabling symptoms.

$$A = \frac{F}{K\sqrt{P_1 - P_2}}$$

where A = cross-sectional area of the valve orifice
F = blood flow through the orifice
K = empirical constant
P_1 = pressure proximal to the stenotic orifice
P_2 = pressure distal to it

It is apparent that stenosis can result in a reduction of blood flow (cardiac output) or an increase of pressure proximal to the obstruction. In aortic, mitral and tricuspid stenosis, most of the patient's disability stems from the high pressure proximal to the obstruction (P_1); in pulmonic stenosis, partly from this but mainly from a reduction in flow. Flow bears a direct relation to orifice size, pressure an inverse square root relation. In cases of aortic stenosis, P_1 is the left ventricular systolic pressure; in mitral stenosis, left atrial and pulmonary pressures; in pulmonic stenosis right ventricular systolic pressure; and in tricuspid stenosis right atrial and systemic

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venous pressure. Because of the square root relationship, there is an inordinate rise of pressure proximal to the stenosis as the orifice becomes critically small. By the same token, slight increases of orifice size made by surgery result in a striking fall of the pressure (P_1) proximal to the stenosis.

Aortic Stenosis

There is considerable controversy regarding the cause of aortic stenosis, but whether it be rheumatic or arteriosclerotic, calcification and gross distortion of the valve are extremely frequent in the older age group. In infancy and childhood, congenital aortic stenosis is not calcific.

Recent work on this lesion has thrown considerable light on its hemodynamics. The cross-sectional area of the normal adult aortic valve orifice is about 3 sq. cm. As a result of disease, this becomes narrower. The pressure in the left ventricle rises and maintains an essentially normal output until almost terminally. The development of stenosis is slowly progressive and as it progresses the left ventricle hypertrophies. The ceiling to which the left ventricular pressure rises appears to be about 300 mm. Hg. Because of this enormous capacity to develop pressure, the patient encounters only minimal disability over the years. There is a critical narrowing, however, which eventuates in the appearance of severe symptoms—angina, syncope, left ventricular failure. In the absence of other cardiac lesions, these symptoms never appear at a valve size that is appreciably larger than 0.7 sq. cm. and often not until it is smaller. The appearance of one or more of this triad of symptoms is prognostically ominous. The average duration of life after their advent is about 2 years, although there is a wide individual variation. Estimation of the severity of aortic stenosis by clinical means other than left heart catheterization has been well-nigh impossible in our experience before the advent of these practically terminal symptoms.

During the years before critical narrowing of the aortic valve, the patient usually limits extreme activity voluntarily. For years there is an aortic systolic murmur, loudest in midsystole, transmitted to the neck, sometimes accompanied by a thrill and a diminished aortic second sound. The loudness and character of the murmur give no clue to the severity of the stenosis. On ECG, changes of left ventricular hypertrophy appear at a fairly early and asymptomatic stage of the dis-

ease. Electrocardiographic changes of old myocardial infarction are extremely important to recognize because they are indicative of complicating coronary sclerosis. The angina of aortic stenosis alone is not accompanied by these changes. Their absence as well as their presence makes it difficult to decide how much of the patient's disability stems from coronary disease and how much from aortic stenosis. Fluoroscopy shows the rounded appearance of hypertrophy of the left ventricle with little increase in over-all heart size until late, when dilatation and failure of the left ventricle produces enlargement of this chamber that can be seen radiologically. The left atrium shows only minimal enlargement. The aortic valve may be calcified.

Venous catheterization throws little light on the severity of this condition, but left heart catheterization pin-points its severity quite accurately. This consists of introducing a needle into the left atrium through a bronchoscope or preferably, in our experience, through the back and threading a small polyethylene catheter through the needle and on through the mitral valve into the left ventricle. A needle is inserted in the brachial artery, whose pressure approximates that in the ascending aorta. In this way, pressure may be recorded proximal and distal to the aortic valve. It is essential to measure cardiac output at the same time because, although output is usually well maintained until terminally in aortic stenosis, it may be low, particularly if there is any associated valvular or myocardial disease. The easiest method of determining flow under these conditions is by the dye dilution method, injecting the dye directly into the left atrium and sampling from the inlying needle in the brachial artery.

The degree of aortic stenosis may be calculated quite accurately by the method of Gorlin and Gorlin:

$$AVA = \frac{AVF}{44.5 \sqrt{LVsem - BAsm}}$$

where AVA = cross-sectional area of the aortic valve, in sq. cm.
AVF = flow through the aortic valve in cc./systolic second = cardiac output in cc./sec. divided by the systolic ejection period in sec./min.
44.5 = empirical constant
LVsem = left ventricular systolic ejection mean pressure, mm. Hg
BAsm = brachial arterial systolic mean pressure, mm. Hg

If aortic regurgitation is present, a falsely low value for valve size will be obtained, but regurgitation can be recog-

nized, at least qualitatively, by the presence of a murmur.

In our experience, the duration of systolic upstroke of the brachial arterial pressure has been of little help in evaluating aortic stenosis, since so many factors other than the stenosis are responsible for its duration. It correlates better with stroke output than with aortic valve size.

The surgical relief of aortic stenosis is fraught with difficulty. The problem is to relieve the stenotic valve obstruction, restore valve action, and at the same time avoid aortic regurgitation. In looking at many of these deformed, calcified valves, one wonders whether it will ever be possible to obtain any sort of valve action from them. Postmortem motion pictures of the action of aortic valves of patients with severe aortic stenosis, by McMillan *et al.*, have been very discouraging to the idea that such valves can ever be restored to function. Numerous reports on aortic stenosis surgery have appeared in the last few years. Operative approach to the valve has been either transventricular or by way of the aorta, with the latter perhaps being a more sound approach since it does not produce the added, poorly tolerated burden of ventricular damage, which is unavoidable when even a small cardiotomy wound is made in the left ventricle.

In published reports, the operative risk has varied between about 15 and 35%, with an over-all figure of perhaps 25%. It must be borne in mind, however, that these patients have as a rule been desperately ill and form a group for whom the usual medical therapy has little to offer.

It is difficult to evaluate the results of surgery. Most of the reports state that a small proportion of patients are clinically much better, but this clinical impression has not been documented by objective measurements of transvalvular pressure gradients and flows. The limiting factor in relieving stenosis is regurgitation. How often this is produced cannot be concluded from the literature. That surgeons themselves are not too enthusiastic about their results is perhaps best illustrated by the not infrequent suggestion that operative intervention be carried out at an earlier stage of the disease, when the valve is more mobile and not completely deformed by rigidity and calcification. With this suggestion I have no traffic, because the prognosis may be excellent for many years to come in such individuals and at earlier stages medical therapy is just as effective as surgical and far less hazardous. The problem which must be faced squarely from a surgical viewpoint is the correction of stenosis at the late stage of the disease

AORTIC STENOSIS
LEFT HEART CATHETERIZATION
PRE OPERATIVE
POST OPERATIVE
LV
254
20
LV
190
50
BA
117
80
BA
120
60
EKG
EKG
105 beats/min.
106 beats/min.
250
200
150
100
50
0
mm.Hg
120
100
80
60
FLOW 6.4 L/min.
GRADIENT (SM) 107 mm.Hg
AVA 0.4 cm.2
FLOW 5.9 L/min.
GRADIENT (SM) 48 mm.Hg
AVA 0.6 cm.2
J.I.

FIG. 1.

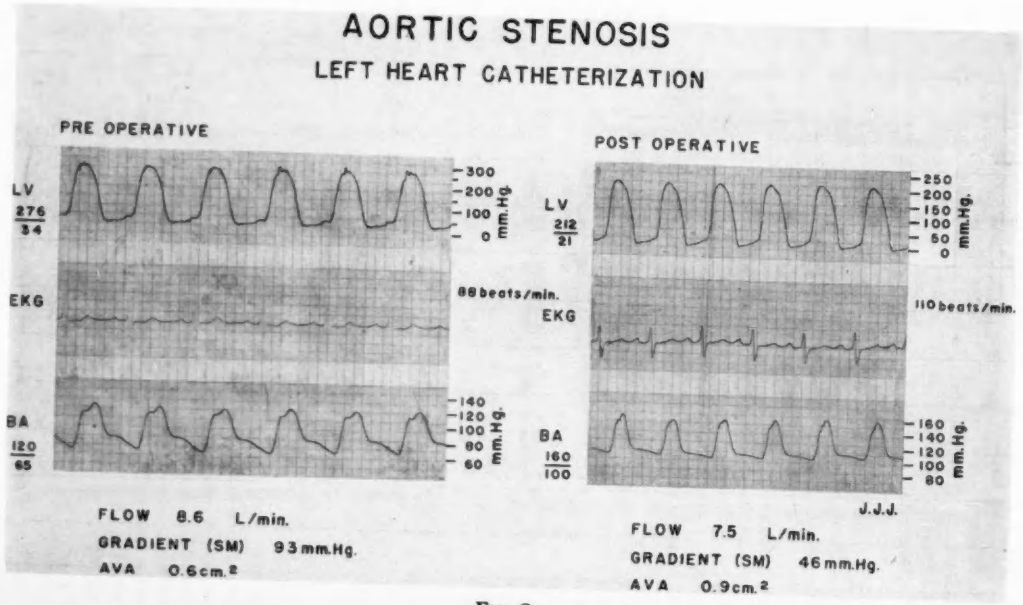
AORTIC STENOSIS
LEFT HEART CATHETERIZATION
PRE OPERATIVE
LV
276
34
300
200
100
0
mm.Hg.
EKG
88 beats/min.
BA
120
65
140
120
100
80
60
mm.Hg.
FLOW 8.6 L/min.
GRADIENT (SM) 93 mm.Hg.
AVA 0.6 cm.²
POST OPERATIVE
LV
212
21
250
200
150
100
50
0
mm.Hg.
EKG
110 beats/min.
BA
160
100
160
140
120
100
80
mm.Hg.
J.J.J.
FLOW 7.5 L/min.
GRADIENT (SM) 46 mm.Hg.
AVA 0.9 cm.²

FIG. 2.

when, in adults, function of the valve is almost destroyed.

For this reason, work is proceeding apace in the use of artificial valves. Such work has not as yet developed to the point of human application. As an illustration of ingenuity and originality might be cited the work of Donovan and Sarnoff, who, in dogs, have created an artificial outlet from the apex of the left ventricle leading through an artificial valve (Hufnagel type) and then to the descending aorta. Blood perfuses the upper half of the aorta in retrograde fashion and the lower half in the usual direction. In these animals, the root of the aorta may be ligated and the animal enjoy full, even violent, activity. Apparently the left ventricle functions admirably, with blood being ejected either from its base (normal situation) or its apex (Donovan-Sarnoff experiment).

At the present stage of surgical effectiveness, it is hard to answer the question as to how patients can be selected for surgical correction of aortic stenosis. Certainly surgery is contraindicated before angina, syncope or left ventricular failure has occurred. But even then, should surgery be performed? I must confess that I do not know the answer. I have elected to withhold surgery in a fair number of patients, most of whom died of their disease within six months. And I have sent patients to surgery who did not survive the procedure or who were little improved clinically or by objective measurements (Fig. 1). On the other hand, an occasional patient will show dramatic improvement clinically as well as objectively (Fig. 2).

Mitral Stenosis

This lesion is rheumatic in origin. The rare case of congenital mitral stenosis will not be discussed. Surgery has given a real impetus to the study of this lesion.

Generally speaking, in patients with the typical murmur of mitral stenosis, disability is on the basis of rheumatic myocarditis if the patient is under the age of 20, and is due to mechanical obstruction at the mitral valve in those over 30. There are, of course, individual exceptions to each statement. Narrowing of the mitral valve is progressive in many but not all patients, and the average age of disability is in the fourth and fifth decades.

As the mitral valve becomes progressively narrower, flow through the valve (cardiac output) decreases moderately;

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the important change is a rise of pressure in the left atrium, which is normally about 5 mm. Hg at rest. Assuming the osmotic pressure of plasma to be 25 mm. Hg, the pressure in the left atrium (and therefore pulmonary veins and capillaries) cannot exceed this value without producing pulmonary edema. Twenty-five mm. Hg or thereabouts is the limit to which this pressure can rise at rest, and generally speaking this occurs at rest when the mitral valve has an area at or near 1.0 sq. cm., which is about ⅕ the normal valve size. The main disability in mitral stenosis is respiratory, due, in the final analysis, to pulmonary congestion which, in turn, is due to the elevated pressure in the pulmonary capillaries. Thus, as the valve becomes narrow, dyspnea first appears only on extreme exertion when the valve area is about 2.0 sq. cm.; with further narrowing of the valve, dyspnea appears with less and less exertion until at a valve area of about 1.0 sq. cm. there is dyspnea at rest or else on the slightest exertion. Orthopnea, attacks of paroxysmal nocturnal dyspnea, and gross hemoptyses are prone to be present at this point, but the only part of the heart that is seen radiologically to be enlarged is the left atrium. The ECG shows no evidence of ventricular hypertrophy.

A second circulatory stenosis appears when, but not before, the mitral valve approaches 1.0 sq. cm. This second "stenosis" is in the pulmonary vasculature. The associated pulmonary vascular disease has a profound effect on the clinical course of the patient. Pressure in the pulmonary artery must rise to maintain flow through the pulmonary vascular as well as the mitral stenotic obstruction. Pulmonary arterial hypertension becomes progressively severe. The main trunk of the pulmonary artery becomes progressively larger; the right ventricle hypertrophies and eventually fails, producing hepatomegaly, venous distention and systemic congestion.

The physical signs of mitral stenosis are the rumbling diastolic murmur with presystolic accentuation if the rhythm is normal sinus, an accentuated apical first sound, usually an opening snap, and an accentuated and split second sound in the pulmonic area. Apical systolic murmurs are often present without necessarily indicating the presence of mitral regurgitation.

The electrocardiographic changes may consist of P-mitrale and right ventricular hypertrophy, but the latter does not occur until there is moderately severe pulmonary vascular disease in addition to severe mitral stenosis.

The radiologic changes of mitral stenosis alone consist, early, of enlargement of the left atrium, usually before the murmur appears and, later, prominence of the hilar vascular markings and at times calcification in the region of the mitral valve. The heart is not enlarged. When pulmonary vascular disease complicates mitral stenosis, there is progressive enlargement of the heart as a whole and specifically of the right ventricle and main pulmonary artery.

Surgery for mitral stenosis is generally accepted to be highly beneficial. Only a few reports express a complete lack of enthusiasm and among these the lack of evidence of objective improvement (cardiac output, pulmonary "capillary" pressure and arterial pressure, calculation of size of mitral valve orifice, and pulmonary vascular resistance pre- and postoperatively) indicates either little or no surgical enlargement of the stenotic mitral valve or, as an alternative possibility, production of mitral regurgitation. The main body of evidence indicates that surgery is truly beneficial for the great majority, both clinically and objectively.

Great improvement occurs in 70 to 80% of cases after surgery. This improvement depends on the stage of disability present before operation. Those with severe mitral stenosis, uncomplicated by pulmonary vascular disease, return to full activity, but few would be able to participate in competitive sports. They would be completely asymptomatic in ordinary daily life, indulging in what might be termed normal middle-aged activity (stair climbing at a normal pace, shopping and carrying bundles, all forms of housework, golf, swimming). Patients who were in group 4 preoperatively, with advanced pulmonary vascular disease, huge failing hearts, and cachexia, do not demonstrate such striking improvement. It may take months for the pulmonary vascular disease to disappear. It may take a year or two for body weight to return to normal and for musculature, wasted by malnutrition and atrophic from disuse, to return to strength again. The large, dilated hearts show but a tendency to return toward normal. These group 4 patients do not have the return of physical activity enjoyed by those operated on at an earlier stage of their disease. On the other hand, about half or more can carry on activities normal for a person in his sixties. The remainder of the group show some improvement, no improvement, or do not survive surgery.

The surgical risk is about 2 to 3% in class 2 and class 3, but in class 4 with complete invalidism, severe pulmonary

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vascular disease, cardiomegaly, and right ventricular failure, the operative mortality is about 20%.

Although most patients are benefited, it is only fair to point out the limitations of surgery. In about 5%, it is technically almost impossible to obtain satisfactory relief of the stenosis, due particularly to subvalvular stenosis from fusion of chordae tendineae on the ventricular side of the mitral valve or to extensive calcification.

In some, mitral regurgitation will be produced on relief of the stenosis. Fortunately, this is largely avoidable, providing the surgeon does not seek relief of stenosis too aggressively. How often regurgitation is produced is impossible to say because of the lack of adequate methods for its detection. The appearance of an apical systolic murmur postoperatively does not necessarily mean that regurgitation has been produced, although in some cases it certainly does mean just that. From our own studies, it appears that the mitral valve cannot be opened above an area of about 1.8 to 2.2 sq. cm. without producing regurgitation of clinical importance. The postoperative exercise tolerance of these patients is, however, in accord with our calculations of postoperative valve size and of postmortem measurements. Thus, the mitral valve orifice is not restored to anywhere near its normal size, but it is usually enlarged to a size that results in dramatic clinical improvement.

Surgery does not render these patients immune to further attacks of rheumatic activity. Indeed, some patients do poorly postoperatively because of this. About 20% develop one or more attacks of what has been called the "post-commissurotomy" syndrome, some of which are clearly a flare-up of rheumatic fever while others are obviously pulmonary embolism or "virus" pneumonia. There is a sizable group, however, whose nature has never been adequately elucidated.

There has been a good deal of speculation with respect to the sealing off again of these valves following surgery. Although there are occasional cases suggesting that this has occurred, there is as yet no convincing evidence to indicate that it has happened on a wide scale. In our opinion, however, there is no reason to believe that the valves will not become narrowed again by the same process at work preoperatively. Only seven years have transpired since the operation was devised and it seems probable that if narrowing of the mitral orifice is indeed occurring, it will not be evident for another three or four years.

With respect to the selection of patients for surgery, it seems proper to wait until either the patient is sufficiently symptomatic (dyspnea on climbing one flight of stairs at a normal pace) or, as is occasionally found, with few symptoms but with cardiac enlargement of 20% or more and right ventricular hypertrophy seen on the electrocardiogram. Irrespective of cardiac dysfunction, arterial embolism is an indication for surgery. Although another may never occur, recurrence of embolism can be disastrous. Surgery is far more effective than any other form of therapy in preventing further arterial embolization. Surgery in patients at an earlier stage of mitral stenosis seems entirely unwarranted. As in the case of asymptomatic aortic stenosis, medical therapy is just as effective as surgical and far less hazardous. Age is no barrier. Although the risk increases with advancing years, patients in their 60's have been greatly benefited. It is preferable to operate between rather than during pregnancies but if one's hand is forced, surgery should proceed as usual.

Pure Pulmonic Stenosis

Pulmonic stenosis is almost invariably congenital in origin; rheumatic involvement of the valve and malignant carcinoids are a rarity. Pulmonic stenosis produces an obstruction to the flow of blood from the right ventricle. The orifice does not become progressively smaller as is the case with stenosis on a rheumatic basis. On the other hand, the pulmonic orifice does not appear to enlarge with the growth of the individual.

There are two forms of pulmonic stenosis—valvular and infundibular. The valvular type consists of a fusion of the commissures with a hole of variable size where they meet in the middle. It resembles an inverted cone with a central aperture at the narrow end. The infundibular type is a narrowing of the outflow tract of the right ventricle beneath the valve. The narrowing is of varying lengths and diameter. The surgical and pathologic anatomy of these two types has been described in detail by Brock. These two types of stenosis may usually be differentiated accurately by cardiac catheterization and it is important that they be distinguished because the problem and effectiveness of surgical correction is different for the two types.

Pulmonic stenosis produces varying degrees of systolic hypertension in the right ventricle and a variable reduction

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of cardiac output, depending on the severity of the lesion. Systolic pressures can rise as high as 200 mm. Hg in the right ventricle. The orifice of a normal adult pulmonic valve in systole is about 5 sq. cm. Disability occurs when the valve area is less than about 0.7 sq. cm.

Although symptoms may appear at any time during life, they are most likely to occur during the rapid-growth periods of infancy and adolescence. Disability stems particularly from low cardiac output and from right ventricular failure. In mild pulmonic stenosis, cardiac output is well maintained with variable increases of right ventricular pressure. Although the cardiac output is usually reduced when the right ventricular systolic pressure exceeds 100 mm. Hg, it may be symptomatically low in certain individuals whose right ventricular systolic pressure is no greater than 65 mm. Hg. A reduced cardiac output leads to fatigue, loss of energy, exertional dyspnea of the noncongestive type, and the violaceous type of stagnant cyanosis so well described by Peacock in 1879. Another cause of cyanosis, occurring especially in the third decade, is a right-to-left shunt through a patent foramen ovale (trilogy of Fallot).

Right ventricular failure occurs only with the most severe stenoses. Syncope, anginoid pain, and sudden death occur only in those with severe pulmonic stenosis.

Physical examination reveals a harsh, diamond-shaped pulmonic systolic murmur, maximal in the second or third left intercostal space, usually accompanied by a thrill and a diminished pulmonic second sound in all but the mildest cases. A sternal heave is apt to be present in those with a valvular type of stenosis and absent in those with an infundibular type.

Electrocardiographic evidence shows that right ventricular hypertrophy is the rule except in cases of extreme mildness.

On radiologic examination, the right ventricle is found to be enlarged; the pulmonary vascular markings are normal or decreased, and the main trunk of the pulmonary artery is often very prominent due to post-stenotic dilatation. This is seen most commonly in the valvular type of pulmonic stenosis, but we have seen it not infrequently in the infundibular type.

The most precise method of evaluation is by cardiac catheterization. Normally there is no measurable systolic pressure gradient between right ventricle and pulmonary artery. In pulmonic stenosis, the presence of a gradient es-

tablishes the diagnosis. When the pressure gradient and flow are known, the pulmonary valve orifice size can be readily calculated with considerable accuracy as follows:

$$PVA = \frac{PVF}{44.5 \sqrt{RVsem - PAsm}}$$

where PVA = cross-sectional area of the pulmonary valve, sq. cm.
PVF = flow of blood across the pulmonary valve during systole = cardiac output (cc./min.) divided by systolic ejection period (sec./min.)
44.5 = empirical constant
RVsem = mean right ventricular systolic ejection pressure, mm. Hg
PAsm = pulmonary arterial mean systolic pressure, mm. Hg

Surgery for pulmonic stenosis was introduced by Brock, using a valvulotome with a transventricular approach. He has met with excellent success in relieving both valvular as well as infundibular stenosis, as indicated by a reduction of the pressure gradient between right ventricle and pulmonary artery. His technic has been widely used. In recent years there has been a trend toward performing this surgery under direct vision, using an extracorporeal pump or by means of hypothermia. It is apparent that surgery for the valvular type of stenosis is extremely beneficial even though some valvular obstruction persists postoperatively, as indicated in most reports by a continuing pressure gradient. This is the one type of stenosis surgery in which the production of regurgitation in exchange for the relief of stenosis is not a limiting factor. Free pulmonic regurgitation is a perfectly benign lesion, providing pulmonary hypertension does not exist.

Surgery for infundibular pulmonic stenosis is, generally speaking, more difficult and less satisfactory in the hands of most surgeons, although Brock's experience has been excellent in this respect.

One group of authors has considered the mere presence of pulmonic stenosis to be an indication for surgery. Since this type of surgery promises to improve as more experience is gained, it does not seem justifiable to operate simply because the lesion exists. It is to be emphasized that in the asymptomatic individual, medical therapy or no therapy is just as efficacious as surgery. Some authors have considered a right ventricular systolic pressure of 70 to 100 mm. Hg to be an indication for surgery. This concept does not appear to this

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author to be entirely sound, since the right ventricular pressure rises and falls with cardiac output as the patient's state changes. Furthermore, certain individuals tolerate well a given degree of stenosis and right ventricular pressure; others do not. Rather than to use an isolated and arbitrary reading of the right ventricular pressure as an indication for surgery, it seems to this author more rational and judicious to reserve operation for those who have untoward symptoms or signs of an enlarging heart, as advocated by Gibson *et al.*, for then surgery has much to offer. Until these complications appear, the prognosis remains excellent.

Tricuspid Stenosis

Tricuspid stenosis occurs in 10 to 15% of patients with chronic rheumatic heart disease and rarely exists as an isolated lesion. The manifestations of this lesion are therefore frequently masked by the associated mitral stenosis. Yu and his coworkers have recently reviewed the literature on its surgical aspects. They point out that this lesion can be suspected by finding (1) a diastolic rumbling murmur in the tricuspid area accentuated by inspiration, (2) a prominent "a" wave in the jugular pulse, (3) P-pulmonale in the ECG, (4) absence of physical signs of marked pulmonary hypertension or of physical or electrocardiographic signs of right ventricular hypertrophy although the patient is markedly disabled, and (5) disproportionate right atrial enlargement visible fluoroscopically. Diagnosis is established by finding a significant diastolic pressure gradient across the tricuspid valve.

Surgical experience in the correction of this lesion is confined to a handful of cases so that satisfactory evaluation of the present status of its surgery cannot be made. As Yu *et al.* emphasize, in a patient with combined mitral and tricuspid stenosis the expected improvement may not occur unless the stenosis of both mitral and tricuspid valves is corrected. As with the aortic and mitral valves, the limiting factor in the relief of tricuspid stenosis is the production of regurgitation.

VALVULAR REGURGITATION

Regurgitation represents a very real and common problem, but there is a dearth of information on its physiology, since methods are not available for its measurement. Gorlin and

Dexter have made a hydraulic analysis of the problem which has thrown considerable light on the factors regulating and affecting its magnitude. Their methods of calculation are admittedly semi-quantitative and are not applicable to a detailed study of the problem. Recently, Korner and Shillingford have shown that the form of a dye curve obtained from the brachial artery after injection of dye into a systemic vein or central circulation has three determinants: cardiac output, blood volume between injection and sampling sites, and regurgitation. Since output and volume can be determined, they have analyzed the down-slope of the curve mathemati-

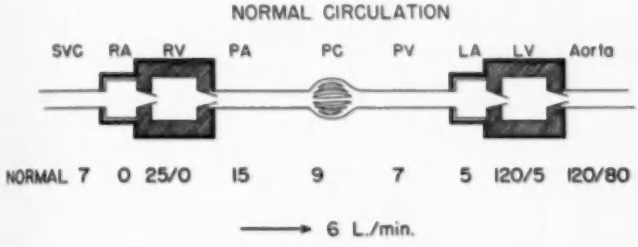

FIG. 3.

cally so as to recognize qualitatively and calculate quantitatively the magnitude of the third factor, regurgitation, if such is present. This method may perhaps allow not only careful analysis of regurgitation physiologically and clinically, but also evaluation of the effectiveness of surgical procedures for its correction.

The same principles apply to regurgitation as to stenosis; i.e., the regurgitant orifice size bears a direct relation to regurgitant flow and an inverse relationship to the square root of the pressure gradient across the regurgitant valve.

A small regurgitant orifice in a high pressure area can lead to the regurgitation of large volumes of blood. This is best seen in aortic regurgitation, where the pressure gradient between the aorta and left ventricle is moderately high; in mitral regurgitation in which the regurgitant gradient between the left ventricle and left atrium in systole is even higher, in pulmonic regurgitation in which the gradient between pulmonary artery and right ventricle in diastole is normally very low; and in tricuspid regurgitation in which

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the gradient between the right ventricle and auricle in systole is of only moderate degree (see Fig. 3).

With an aortic regurgitant orifice of a given size, it is obvious that diastolic hypertension in the aorta increases the volume of blood regurgitated, whereas a lowering of aortic diastolic pressure or a rise of left ventricular diastolic pressure (left ventricular failure) decreases the regurgitant flow. Systolic hypertension increases the volume of blood regurgitated in mitral insufficiency; pulmonary hypertension, systolic and diastolic, increases pulmonic and tricuspid regurgitation.

Regurgitation results in an increased total ventricular output but a decrease in the amount of blood that is impelled forward. From the cardiac viewpoint, it is a high output state; from the point of view of what the body receives, low output.

There are two principal anatomic derangements involved in regurgitation and many secondary features. The first is absolute loss of substance; i.e., the valve is deformed so that it does not close properly with a tight seal. This condition may be due to scarring, calcification, shortening, contraction, or rupture of leaflets and chordae tendineae. The second is relative loss of substance due to enlargement of the valve annulus because of ventricular dilatation (functional insufficiency).

Both absolute and relative loss of substance are frequently combined because, as the ventricle in question dilates as it so frequently does, the annulus dilates, producing a larger regurgitant valve area. This in turn leads to more regurgitation, more dilatation, and greater regurgitant area: a vicious circle has been established.

Aortic Regurgitation

This may be due to rheumatic heart disease, syphilis, bacterial endocarditis, trauma, or a congenital lesion.

In aortic regurgitation, the left ventricle pumps into the aorta a larger than normal amount of blood, some of which leaks back into the left ventricle. The double blood flow into the left ventricle during diastole from both aorta and left atrium produces dilatation of this chamber and eventually hypertrophy. The largest hearts on record have been due to this lesion. Little information is available with respect to the precise hemodynamic abnormalities. The most precise analy-

sis to appear to date was recently reported by Gorlin *et al.*

The course of the disease is variable. There may be no disability for years. On the other hand, disability may occur in childhood if the lesion is sufficiently severe. Headaches and faintness on rising suddenly or on effort are early symptoms. True angina pectoris or anginal-like pain are frequent. Exertional dyspnea is likewise an early symptom and may progress rapidly and intractably to frank congestive heart failure.

On physical examination, the diagnostic murmur is a high-pitched diminuendo diastolic murmur, heard at the base and loudest along the left sternal border. An accompanying aortic systolic murmur may or may not denote an associated stenosis of the aortic valve. A_2 may be ringing, normal, or absent.

The left ventricle enlarges early, extending downward and outward so that the apex impulse becomes maximal in the sixth intercostal space and out toward the axilla. Since dilatation of the left ventricle plays such a part in aortic regurgitation, the impulse eventually becomes diffuse and heaving rather than discrete and thrusting, as in the case of the hypertrophied left ventricle of aortic stenosis.

The character of the pulse is important. It is best felt over the carotids, which are nearer the "source of supply" than over the more distant radials. As aortic regurgitation increases in severity, the pulse becomes Corrigan in nature. The systolic pressure rises, the diastolic falls, and pistol shots, Duroziez's sign, capillary pulse and the like make their appearance.

Left ventricular hypertrophy demonstrable by ECG occurs early in the course of aortic regurgitation.

Fluoroscopy reveals enlargement of the left ventricle early and its size is roughly proportional to the severity of the aortic regurgitation. The left atrium remains normal in size or at most minimally enlarged. A "rocking beat" appears. As the left ventricle fails, pulmonary vascular congestion becomes apparent.

Surgery for this lesion has been attempted by Hufnagel by inserting an artificial valve into the aorta just distal to the branch of the left subclavian artery. Thus, there is no regurgitation below this point but it persists proximal to the valve. Hufnagel's surgical mortality has been about 6% and the hospital mortality 20% in 80 patients, a risk that is reasonable considering the fact that patients with severe aortic disease are among the worst surgical risks and that these

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were all class 4 cardiacs. The benefit to be derived from this type of surgery is not entirely clear. Of 23 patients reported by Hufnagel *et al.* in 1954, there were 6 deaths and all of the 17 survivors followed up to 11 months after operation were better. Our own experience has been small (6 cases) but has not been encouraging from the viewpoint of left ventricular function and postoperative clinical improvement. McKusick *et al.*, studying the circulation in a model, concluded that the benefit obtained from this operation represented a balance between a decrease of cardiac work and a decrease of coronary blood flow due to a reduction in the size of the reservoir proximal to the artificial valve. How close to the coronary orifices the valve can be placed without interfering with coronary flow in man, whose capacity to develop circulatory compensations is so different from that of a model, is not known. For technical reasons, placement of the valve in the arch of the aorta is not feasible. The ideal location for such a valve would be in the left ventricle or aorta, proximal to the coronary ostia. So far, placing it there has not been possible.

Such surgery represents a step in the surgical correction of this common and serious disease, but it represents a compromise. There is no justification for its application in any but the most seriously ill patients with this lesion.

Another approach is constriction of the aortic ring and use of various types of pericardial and prosthetic valves to allow the valve leaflets to approximate one another more closely in diastole. There is too little information available to form an opinion with respect to its effectiveness.

Mitral Regurgitation

Mitral regurgitation has received increasing attention in recent years. Although pure mitral stenosis and pure mitral regurgitation are easily recognized, moderate degrees of each may co-exist; their relative severity is then hard to assess.

In mitral regurgitation, two orifices compete for the output of the left ventricle—the aorta and the regurgitant mitral orifice. Some blood is ejected into the aorta and some back into the left atrium. The total left ventricular stroke output is larger than normal; the amount going out the aorta is less than normal. The blood regurgitated into the left atrium produces a momentary rise in the pressure in the left atrium during ventricular systole (elevated V-wave) but even this is inconstant. Its effect on the mean left atrial pressure is very

slight. Pulmonary hypertension is not produced by the regurgitation itself. Depending on the severity of the regurgitation, left ventricular failure may eventually appear and its diastolic pressure then rises, producing pulmonary congestion. The right ventricle likewise dilates and fails soon after dilatation of the left ventricle. Before left ventricular failure has occurred, the patient may become incapacitated by easy fatigability and loss of energy and ambition due to the reduced flow of blood out of the aorta. When the diastolic pressure rises in the left ventricle, all the manifestations of left ventricular failure occur and now the patient complains of shortness of breath and not of easy tiring. When cardiac dyspnea and fatigue co-exist, the patient complains only of dyspnea. The course may be rapidly downhill or prolonged, as is the case in other types of left ventricular failure. If the course is prolonged, pulmonary vascular disease may occur, similar to that seen in mitral stenosis but not so severe.

The salient physical signs of mitral regurgitation are an apical, pansystolic murmur transmitted to the axilla. Many such murmurs of grade III intensity or less are functional; i.e., are not a mark of mitral regurgitation. If the murmur is grade IV or more, it almost always indicates the presence of mitral insufficiency. We have observed six cases of severe, incapacitating mitral regurgitation, verified at operation, without a murmur. The first sound at the apex is rarely accentuated. An opening snap is rarely present. We have observed it in three cases of free mitral insufficiency, confirmed by sound tracing, with the regurgitation verified at operation. Other physical signs are concerned with evidence of left ventricular enlargement. It has been reported that a sternal heave is a sign of advanced mitral stenosis. This is not the case, however, because the right ventricle becomes dilated and hypertrophied along with dilatation and hypertrophy of the left ventricle in many cases of mitral regurgitation. We have observed a sternal and parasternal heave in many of our insufficiency cases.

The ECG reveals either no hypertrophy or left ventricular hypertrophy. There are rare exceptions. In over 1,000 patients with mitral disease personally observed, about 12 with free regurgitation have had right ventricular hypertrophy as shown by ECG. In each, there was evidence of moderately severe pulmonary vascular disease and pulmonary hypertension to which we attributed the right ventricular hypertrophy.

There is usually little difficulty in differentiating pure

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mitral stenosis from pure mitral regurgitation. Severe stenosis and severe regurgitation are mutually exclusive. On the other hand, moderate degrees of each can co-exist, and it is here that much diagnostic confusion arises, because it is truly a mixed lesion. In mitral regurgitation, the total left ventricular output is increased. The ventricle fills during diastole and, if there is some stenotic narrowing of the mitral valve, there will be obstruction to the large flow of blood into the left ventricle during diastole. Mild to moderate anatomic

TABLE 1.

	Normal	A.L. Severe Pure Mitral Stenosis	M.C. Mixed Mitral Stenosis and Mitral Insufficiency
Total left ventricular output, L./min.	6.0	4.1	9.3
Aortic output, L./min.	6.0	4.1	2.6
Regurgitant flow, L./min.*	0	0	6.7
Left ventricular diastolic pressure, mm. Hg	5	4	10
Left atrial mean pressure, mm. Hg	5	26	24
Mitral valve gradient of pressure, mm. Hg	0	22	14
Mitral stenosis orifice size, sq. cm.	5	0.7	2.4

* Calculated by method of Korner and Shillingford in the mitral regurgitation case.

stenosis, of no significance by itself, can become of great significance when the left ventricular inflow is large as a result of regurgitation, as was the case in patient M.C. in Table 1. The severe degree of mitral regurgitation entailed a left ventricular output of 9.3 L./min. For 9.3 L./min. of blood to cross the slightly stenotic (2.4 sq. cm.) mitral valve during diastole, the pressure head in the left atrium had to be 24 mm. Hg. Simple calculation, however, indicates that if there were no mitral stenosis (valve size 5.0 sq. cm.), pressure in the left atrium would have to be only 10 mm. Hg; i.e., the same as in the left ventricle in diastole. It might be thought that relief of the mild stenosis would relieve the patient of elevated pressures (24 mm. Hg) in the lung and therefore of her respiratory distress. This might indeed accrue, but the patient would remain completely incapacitated

by the mitral regurgitation. On the other hand, if mitral regurgitation were relieved, the patient would to all intents and purposes be cured. The residual mitral stenosis would be of no clinical significance.

Many ingenious measures have been devised for the elucidation of the nature of the lesion in cases of mixed stenosis and regurgitation. They include the systolic expansion of the left atrium as viewed radiologically, angiocardiography, the height or character of the run-off of the v-wave in left atrial or pulmonary "capillary" tracing, the esophageal pulse wave, the duration of period between the R-wave on the ECG and first heart sound, and the like. None of these has turned out to be definitive because they are at best only qualitative and often not even that. A more direct approach has been by left heart catheterization but, as seen in Table 1, measurement of the pressure gradient across the mitral valve is in itself of little importance unless the flow across the valve is likewise known. The ordinary cardiac output methods do not suffice because they measure only that portion of the left ventricular output that goes out the aorta. We have recently used the method of Korner and Shillingford for calculating regurgitant and total left ventricular flows combined with the calculation of valve orifice size by the method of Gorlin and Gorlin and have so far found consistent agreement with operative findings.

The physiopathology of mitral regurgitation from a surgical viewpoint has been ably described by several workers.

Attempts have been made to replace valve substance or correct retraction of the cusps in experimental animals and in man by a variety of ingenious technics and to reduce the size of the mitral valve ring by circumferential suture.

Little can be said about mitral regurgitation surgery. Results have at best been indifferent. In no report has the author waxed enthusiastic. The variety of procedures that continue to be devised attest to the unencouraging results with previous procedures.

This is a disorder of considerable magnitude with respect to frequency and disability. Since left ventricular pressure during systole is so high, a very small (1–1.5 sq. cm.) orifice will allow perhaps 4 L. or more of blood per minute to regurgitate back into the left atrium. And yet its correction surgically is unbelievably difficult. Perhaps open heart surgery will yield more fruitful results.

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Pulmonary Regurgitation

Pulmonary regurgitation is usually secondary to pulmonary hypertension or to surgery. Rheumatic involvement of this valve is excessively rare. The Graham-Steell murmur of pulmonary regurgitation in cases of mitral stenosis with pulmonary hypertension is thought not to have much clinical significance.

In the absence of pulmonary hypertension, pulmonary regurgitation is of no clinical significance. Blount *et al.* reported that a murmur of pulmonary insufficiency appeared in a number of cases of pulmonic stenosis after surgery under direct vision with hypothermia with restoration of the right ventricular systolic pressure to normal. They considered the pulmonary regurgitation to be of no clinical importance.

If pulmonary hypertension exists, pulmonary regurgitation can be severe and as serious as aortic regurgitation.

Therapy is twofold: (1) Surgical alleviation of pulmonary hypertension where possible (mitral stenosis, patent ductus arteriosus, atrial septal defect, ventricular septal defect); (2) supportive treatment for right ventricular failure (digitalis, low salt diet, diuretics). Direct surgical attack for pulmonary regurgitation is not needed if there is no pulmonary hypertension and, if such is present, the lesion causing the pulmonary hypertension should be the object of correction if possible. Bailey *et al.*, however, have produced constriction of the pulmonic ring for pulmonary incompetence.

Tricuspid Regurgitation

Regurgitation of this valve may be organic or functional. When it is organic, it is almost always due to rheumatic fever. It is rarely an isolated lesion. It is almost always associated with mitral valvular disease and often with aortic disease as well. Functional tricuspid regurgitation is secondary to dilatation of the right ventricle, usually as a result of pulmonary hypertension of severe degree.

The clinical picture of tricuspid regurgitation is often obscured by the associated valvular disability. The right ventricle dilates due to its increased diastolic filling and hypertrophies due to its increased work load. One of the earliest physical signs to appear is an increase in the intensity of the systolic murmur in the tricuspid area on deep inspiration (Ribero-Carvallo). Pressure curves recorded from the right

atrium may show a lack of the descent of the c-wave but only occasionally a large v-wave because it is damped by the large venous volume into which the blood is regurgitated. As regurgitation increases, abnormally large systolic pulsations may be observed in the neck veins and even in the veins of the forearm. The liver pulsates. These latter changes are found only in severe tricuspid insufficiency. As the right ventricle fails, its diastolic pressure rises, as does that in the venous system.

If tricuspid regurgitation is associated with mitral stenosis, surgical relief of mitral stenosis always reduces tricuspid regurgitation because of the fall of pressure in the pulmonary circuit and in the right ventricle during systole. As pressure falls in the right ventricle, the chamber is less dilated so that it seems reasonable to conclude that the regurgitant orifice becomes smaller. The degree of improvement in tricuspid regurgitation following mitral stenosis surgery thus depends on the magnitude of fall of pulmonary pressure.

Organic tricuspid regurgitation can be an incapacitating lesion even in the absence of pulmonary and right ventricular hypertension. There is practically no experience in the surgical correction of this lesion.

CORONARY ARTERY DISEASE

Surgery for coronary artery disease has been based on three general objectives. The first has been directed toward reduction of the metabolic rate by total thyroidectomy and more recently by the use of radioactive iodine. These procedures do not seek to improve the blood supply to the ischemic heart but rather to reduce the cardiac work load. The second has been relief of anginal pain by sympathectomy, introduced by Jonnesco and practiced extensively and often, but inconsistently, successfully by many. The third involves attempts to increase the failing myocardial blood supply. It has had a long and varied history, with three major approaches:

1) Development of vascular adhesions between myocardium and other structures, mainly the pericardium. These various procedures have apparently had a salutory effect clinically. Burchell however, reported that the epicardium constitutes a barrier to the development of any but the smallest collateral blood vessels, i.e., of less than 40 μ. The only method described to date which will destroy this epi-

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cardial barrier without injuring the myocardium is phenol which, as applied clinically, has a low operative risk and a high incidence of relief of pain.

2) A second approach has been arterialization of the coronary veins by producing an arterial shunt from the aorta to the coronary sinus. At a second operation, the coronary sinus has been ligated between the right atrium and the arterial anastomosis. Even though patients are reportedly improved, atheromatous plaques form in the coronary veins adjacent to the arterial anastomosis, there is a tendency to thrombosis of the anastomotic shunt, and evidence suggests that the capillary circulation of the myocardium is increased for only a few months and it is not entirely clear as to whether or not intercoronary collaterals develop.

3) The third approach has been to increase the collateral circulation to the myocardium by means of vascular grafts, particularly the recent development of implantation of the mammary artery into the left ventricular myocardium. The internal mammary artery is implanted into the wall of the left ventricle and branching out of new vessels communicating with the existing coronary circulation has been demonstrated. Vineberg has shown that the dog heart can be supplied through these vessels alone. Others have had difficulty in repeating this work and have found thrombosis or a non-functioning mammary artery in a certain percentage of cases.

There is no disorder more difficult to evaluate subjectively or objectively than angina pectoris and coronary artery disease. Evaluation of surgical procedures in dogs, which are the species in which almost all experimental coronary surgery is performed, is difficult to translate in human terms because of the dog's great ability, in contrast to man, to develop a collateral circulation, be it in the leg, the aorta, the portal system, or by analogy, the heart. Even in the dog, however, evaluation of the efficacy of the various procedures is at best difficult. Vineberg has recently reviewed this methodology. But if it is difficult in the dog, how much more difficult it is in man.

Almost all reports on the efficacy of one or another form of surgery for coronary artery disease have emphasized the symptomatic benefit that the patient has gained. There are really two points to bear in mind; one is the relief of angina pectoris, which in some individuals entails a life of continual pain of a most unpleasant kind. Cardiac denervation, either

preganglionic or postganglionic, can often relieve the patient's suffering at a risk of 10% or less.

The other, and by far the most important, is an increased blood supply to the myocardium. Whether or not this is being accomplished by current surgical technics I do not know. Methods do not suffice to give the answer one way or the other. Proof that revascularization does not take place is no stronger than proof that it does. In the last analysis, the burden of proof must rest on those who say it does, but in the meantime I am forced to maintain a neutral position, giving as strong moral backing as possible to this type of approach because of the magnitude of the coronary disease problem. I myself reserve surgery for relief of status anginosus and if perchance the coronary blood supply increases too, so much the better.

EPILOGUE

Cardiac surgery has had a profound effect on the medical aspects of cardiology. Indeed, it is the surgeons who have given greater impetus to the field of cardiology than anything in the past. If the surgeon is to apply his therapeutic skills to their maxima, he must be armed with the correct diagnosis preoperatively. Not only is qualitative precision a necessity, but it must be quantitative as well. Previously, qualitative diagnosis sufficed. A diagnosis of mitral stenosis and insufficiency was formerly made on the basis of an apical systolic and diastolic murmur. Such a diagnosis is of no help to the surgeon who wishes to know whether the patient's disability is due to mitral stenosis or mitral insufficiency. Will his operation, designed to relieve mitral stenosis, benefit the patient?

Until such time as surgery becomes so perfected that operation will be performed on all patients with a surgically correctible lesion, irrespective of its severity, qualitative and quantitative diagnostic precision is a necessity. This need for quantitative as well as qualitative accuracy of diagnosis has led to the application or development of special technics such as venous catheterization, arterial catheterization, left heart catheterization, angiocardiography in one plane or two planes simultaneously, various methods for measuring cardiac output, including the standard Fick procedure and dye-dilution methods employing dyes or radioisotopes and recording by multiple sampling, cuvettes, oximeters, or well-

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counters. Methods have been devised for measurement of regurgitant flow, calculation of stenotic and regurgitant sizes of valve orifices, location of septal defects by dye dilution curves, and the like. These and many other technics of study have been devised largely to throw light on the condition of the patient so that the surgeon may perform his function armed ahead of time with as detailed information as is possible to obtain. In this way he does his job the best.

Although the surgeon may be thought of as a super-technician by some, it is only too apparent that he knows many facets of cardiology better than does his medical colleague and that, in the years to come, the medical man must work hard to keep up with his surgical confrere. The moral here is that each has a great deal to contribute to the other. Teamwork and an understanding of each other's problems are essential. The best surgeon is a good medical man; but a medical man can never be a surgeon. It is best that the surgeon be relieved of as much of the time-consuming details of the medical work-up as is compatible with his best surgical performance so that he can devote his time and energy to the most highly specialized and skillful cardiologic technics that exist—the operation itself—and depend on the team to help him to this end. The bare essentials of the team consist of the medical cardiologist, radiologist, anesthesiologist, and surgeon. Many other disciplines must be brought into the picture as problems arise. Thus it is teamwork which now and in the future will yield the best results, and this can be obtained only through mutual understanding and confidence. It is a challenge to both the medical man and the surgeon.

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